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M E M O R A N D U M

AUG 15 1978

TO: All Interested Parties

FROM: Michael D. Roach, Chief, Air Quality Bureau

SUBJECT: Ambient Air Quality Standards

DATE: May 17, 1978

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Attached is Working Paper Number 2 as developed by the Montana Ambient Air Quality Study (MAAQS). This paper, and others still to be developed for other pollutant groups, are intended to inform the public and other interested parties as to the scientific evidence on the interactions of pollutants with various aspects of the environment.

Although MAAQS has attempted to review all relevant data, there is a possibility that valid information somehow has not been considered. Part of the purpose of circulating this paper is to ask persons concerned to point out such gaps in the literature or shortcomings in the analyses.

Any new information provided by the public will be considered in the formulation of the basis for setting lead and beryllium standards. In the process of setting standards, economic and environmental issues will be considered along with the scientific data from the updated working paper.

All this information together, plus that generated for all the other pollutant groups, will be issued as a draft environmental impact statement (EIS) in October. An adequate period for public comment will follow.

As it now stands, the working paper is considered a draft, and may have some rough spots. The research results, however, are considered valid and representative of the body of scientific knowledge related to lead and beryllium pollution.

We invite comments and suggestions you might have to improve the quality of this paper.

It should be noted that working papers are behind the schedule presented in Working Paper Number 1. Efforts will be made to meet the schedule in future weeks but not at the expense of scientific validity in subsequent working papers.

We ask that your comments on Working Paper Number 2 get to us by June 5.

JUL 24 1951

Montana Ambient Air Quality Study

DRAFT

MAY 1 1978

LEAD AND BERYLLIUM

Working Paper Number Two for Developing
Enforceable Ambient Air Quality Standards
for Montana

State of Montana
Department of Health and Environmental Sciences
Environmental Sciences Division
AIR QUALITY BUREAU

May, 1978



GLOSSARY

The staff has made every effort to eliminate jargon and technical terms from this paper. Nevertheless, it has been necessary to use some such terms which are not easily translatable into everyday language. Therefore, readers may want to refer to this glossary.

DRAFT

MAY 1 1968

aerosol...system in which the dispersion medium is a gas and the dispersed phase--composed of solid particles or liquid droplets--does not settle out under the influence of gravity

ALAD inhibition....suppression of the ALAD enzyme in the biosynthesis of hemoglobin

aminoaciduria...an abnormal condition characterized by amino acids in the urine

anemia...a reduction below normal in the number of erythrocytes in the blood, reduction in the hemoglobin, and volume of packed red blood cells, resulting in a decrease in the oxygen carrying capacity of the blood.

arteriosclerotic...characterized by a thickening and loss of elasticity of the walls of the arteries

asymptomatic...without symptoms

cardiovascular...of the heart and blood vessel as a unified body system

cognitive defects (CNS)...failure of sight, smell, hearing, or other cognitive modes

coproporphyrin elevation...a greater than normal concentration in the body of an intermediate in the biosynthesis of hemoglobin

electrophysical...having to do with the electrical properties of the body especially nerve activity

encephalopathy...degenerative disease of the brain

endocrine...designating or of any gland producing one or more internal secretions that are introduced directly into the bloodstream and carried to other parts of the body whose functions they regulate or control; any such gland or its secretions, such as the thyroid, pituitary and adrenal glands

erythrocytes...a red blood corpuscle, containing hemoglobin, which carries oxygen to body tissues

fibrosis...and abnormal increase in the amount of fibrous connective tissue in an organ, part, or tissue

free erythrocyte porphyrin elevation...greater than normal concentration in the body of porphyrin, the basis of respiratory pigments.

gastrointestinal...of the stomach and intestines

glomerular atrophy...shrinkage of the glomerulus, the coils of blood vessels vital to kidney function

glycosuria...presence in the urine of glucose, a simple sugar formed from more complex sugars and normally retained in the body as a source of energy

hematopoietic...system of cells in bone marrow, spleen, and lymph nodes concerned with formation of cellular elements of the blood

hemoglobin...the red coloring material of the red corpuscles; it carries oxygen from the lungs to the tissues, and carbon dioxide from the tissues to the lungs.

hepatic...of or affecting the liver

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MAY 1 1978

hyaline degeneration...a form in which there is produced a clear structureless or homogenous translucent change in tissues or cells

hyperphosphaturia...a condition of above normal amounts of phosphate compounds in the urine

immunological...of the branch of medicine dealing with antigens and antibodies

increased urinary ALA excretion...a system of enzymatic inhibition by lead

mitochondrial...of the mitochondria, small organelles found in the cytoplasm of cells; principal sites of generation of energy, they contain enzymes of the Krebs and fatty acid cycles and the respiratory pathway

morphology...form and structure, as of an organism, regarded as a whole

peripheral neuropathies...functional disturbances and/or pathological changes in nerve cells near the outside of the body

placental barrier...a barrier between a fetus and the mother that allows the passage of essential nutrients while protecting the fetus from harmful substances

sorption...absorption or adsorption

Unit Measures

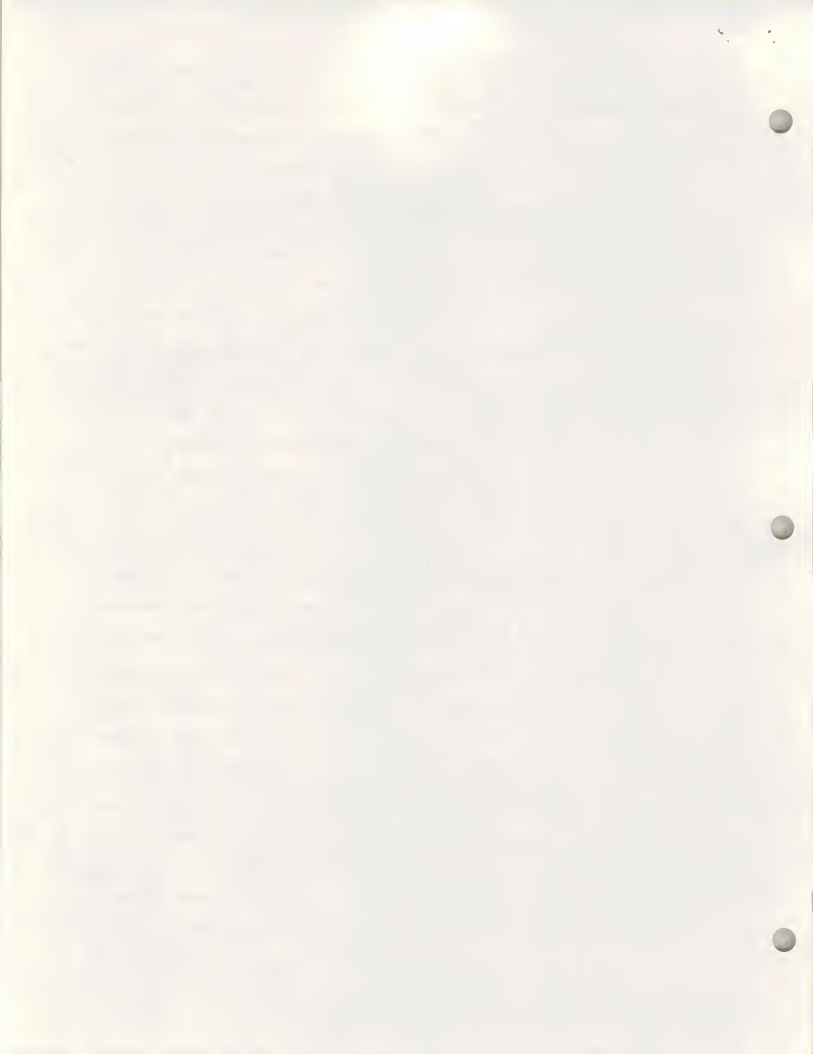
meter.39.37 inches
kilometer (km) 1000 meters, .6213 miles
centimeter.2.54 inches/(1/100th) of a meter
liter.1.057 quarts
deciliter (dl).one/tenth liter or 100 milliliters
milliliter (ml) one/thousandth of a liter, or one/hundreth deciliter
gram(g) 1/454th of a pound, 1/1000th of a kilogram
kilogram (kg) 2.2 pounds, one thousand grams
microgram (ug) 1/1000th of a gram
1 metric ton= 2205 pounds = 1000 kg

Unit Measure Symbols

ug/m³ micrograms per cubic meter
ug/dl micrograms per deciliter

DRAFT

MAY 1 1973



INTRODUCTION & SUMMARY

Beryllium is a light-weight, gray metal used in the manufacture of metal alloys and for a variety of technological applications. Both the metal and its compounds are harmful to human health if inhaled or brought into contact with the skin. Shapley (1977) reports that the National Institute for Occupational Safety and Health (NIOSH) is considering setting a new, more stringent occupational standard for beryllium because some data indicates that beryllium may be a carcinogen.

Although the rationale for setting occupational standards may differ from the reasons for setting ambient standards, it is useful to review occupational standards and data on human exposure to occupationally generated pollutants. One reason is that occupational exposure has, historically, been the source of human dose response data for some pollutants. Factory workers have been exposed to compounds harmful to health and have shown both chronic and acute symptoms.

By reviewing whether occupational standards have been successful in protecting workers, one can get some idea as to what threshold or safe concentrations might be. Based on safe levels of occupational exposure thus determined, an ambient air standard could be set by establishing a margin of safety (setting the ambient standard lower than the occupational standard) based on the assumption that ambient levels may be in the environment 24 hours per day whereas a worker may be exposed for 8 or fewer hours per day.

Currently, the federal emission standard for beryllium is 10 grams of beryllium over a 24 hour period or, as an alternative, an ambient concentration of 0.01 micrograms per cubic meter of air, averaged over a 30 day period. (NIOSH, 1972). There is little or no data from Montana on ambient levels of beryllium in the environment. Also, there is little or no data on health effects of beryllium at concentrations close to the federal standard.

Based on the paucity of data and seeming lack of a serious beryllium problem in Montana, there appears to be little justification for changing the standard from 0.01 micrograms per cubic meter.

BERYLLIUM POLLUTION IN MONTANA

DRAFT

MAY 1, 1978

A. Sources and Emissions

The potential for human exposure to industrially generated beryllium compounds developed in the 1940s as a result of the increased demands for a tough, lightweight, heat resistant metal for use in war materials. Since then beryllium use has expanded and it is a component of X-ray tubes and other electronic parts, metal alloys and nuclear reactors. Until a few years ago, beryllium was also used as a missile fuel and hardener in gas lantern mantles. From 1962-1967, beryllium was processed at Anaconda, Montana. After the processing was terminated in Anaconda, beryllium wastes were sealed in containers and buried in tailings piles (Kelly, 1978). Currently, the potential for elevated levels of airborne beryllium in the Montana environment appears to come from coal fired generating facilities. Abel, et al (1974) reported that the average beryllium concentration in coal was measured at 1-2 parts per million. The Montana Department of Natural Resources (Energy Planning Division, 1974) predicated that for Colstrip generating facilities (plant #1 and #2), emission rates from the stacks would be .0019 grams per second. These values were not however checked for accuracy after the plants become operational. Based on the 24 hour emission rate of .0019 grams per second, 164.16 grams per day would be emitted.

Battelle (1974) estimated that at a coal combustion rate 585,300 pounds per hour, 0.17 pounds per day would escape in the gaseous form. Although emission levels of beryllium compounds have been predicted, it appears that very few monitoring or ambient data have been collected for any potential beryllium source in Montana.

HUMAN HEALTH EFFECTS

The inhalation of beryllium compounds causes respiratory symptoms, the severity of which depends on the nature and duration of exposure. A brief intense exposure may cause chemical pneumonitis and pulmonary edema whereas chronic exposure may cause heart or lung failure. According to Lehman,et al (1966) the pathogenesis of chronic beryllium disease is not well understood. Because not all people exposed to beryllium contract beryllium disease, it was hypothesized that susceptibility was related to individual differences in metabolism.

DRAFT MAY 15 1978

In a document produced by NIOSH (1972), the carcinogenic potential of beryllium was discussed (P.IV-9) and it was reported that compounds of beryllium produced cancer in rabbits following intravenous injection and inhalation. It was further reported, however, that there was no evidence that exposure to beryllium caused cancer in humans. Reeves,et al (1967) exposed 150 rats for 13 months to beryllium sulfate aerosol at concentrations of 21-42 micrograms/cubic meter and produced a 100 percent incidence of cancer. At lower concentrations (2.8 micrograms/cubic meter), over half the rats exposed developed lung cancer.

There is no doubt that beryllium exposure through inhalation produces cancer in experimental animals. An exactly parallel response does not seem to exist for humans however. The following statement (NIOSH, 1972 p.IV-18) summarizes the response differences between animals and man: "the carcinogenic exposure-effect relationship observed in animal does not correlate to man. The human organism has not been observed to respond in the same manner as rats to beryllium exposure; therefore, animal studies contribute only indirectly and provide no correlation of human exposure-effect relationships as they pertain to development of a recommended environmental standard."

ENVIRONMENTAL EFFECTS

A. Odors and Visibility

Airborne beryllium compounds in the Montana environment are too diffuse to be detected by sight or smell.

B. Agriculture and Plants

According to the Community Air Quality Guide for Beryllium (1968), no harmful effects to vegetation from ambient air concentrations have been reported. However, some experimental work shows beryllium to be extremely toxic to bush beans (Gregory, 1964). It not only was toxic but also decreased the copper content of the plant.

C. Livestock and other animals

With experimental animals it has been shown that severe respiratory and cardiac disease and cancer are caused by beryllium inhalation. Such experimental data is not available for livestock and wildlife.

D. Ecosystems

A study in the Powder River Basin in Wyoming of trace metal variation in soils and sagebrush around the Dave Johnston Power Plant dealt with beryllium accumulation due to airborne emissions (Conner, et al 1976). Of the 28 trace metals studied, strontium, vanadium, uranium and selenium showed statistically significant concentration increases closer to the plant. Beryllium, however, showed no change in concentration near the plant.

E. Materials

No reports of harmful effects to materials from beryllium exposure were found in the literature.

POLLUTION CONTROL TECHNOLOGY

A study conducted by Battelle Laboratory (1974) for the Montana Department of Natural Resources assessed the emission control technology for sulfur dioxide, particulates and trace elements in flue gas from large coal fired generating plants. Fabric filtration, electrostatic precipitation and venturi scrubbing were assessed for removal efficiency of particulates. It was found that 50% removal efficiency of gases could be accomplished by venturi scrubbing, the most efficient of the three pollution control techniques assessed for gas removal.

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STANDARDS ADOPTED BY OTHERS

Currently, the national emission standard for beryllium requires that concentrations shall not exceed 10 grams per 24 hour day or total emissions to the atmosphere shall not cause an ambient air concentration above 0.01 micrograms per cubic meter of air, averaged over a 30 day period. The occupational exposure standard is 2 micrograms per cubic meter. In 1975, however, NIOSH proposed lowering the standard to 0.5 micrograms per cubic meter. The World Health Organization in conjunction with the International Labor Organization recommended an occupational exposure concentration of 1.0 - 2.0 micrograms per cubic meter. Other standards are listed in Appendix A-2.

CONCLUSIONS

The current national ambient air standard appears to be adequate to safeguard human health and protect the environment. Based on studies of non-occupational cases of beryllium disease in the human population around a beryllium production plant, the lowest concentration which produced the disease was greater than 0.01 micrograms per cubic meter and probably less than 0.10 micrograms per cubic meter (Eisenbud, 1961). The current federal standard for

ambient air concentrations, 0.01 micrograms per cubic meter, is lower than concentrations observed to cause beryllium disease in humans.

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APPENDIX A-1

MEASUREMENT TECHNIQUES FOR BERYLLIUM

Beryllium

Method: Health Laboratory Science, Vol. 12, No.4, Oct. 1975, pp.383-393

Principle: Ambient air suspended particulate matter is collected on a low background filter medium using a 24-hour sampling interval. Beryllium in the particulate matter is solubilized by nitric acid extraction, facilitated by ultrasonication and heat. The beryllium content of the sampling is analyzed by atomic absorption spectrometry using a nitric oxide/acetylene flame under optimum instrumental conditions at the 234.9 nm absorption line.

Montana's AQB also will recognize any analytical method with a consistent and repeatable lower detectable limit (LDL) of 0.01 Microgram per milliliter of solution. However, the LDL must be documented and presented to the AQB for acceptance. The filter medium must be low in beryllium background and as such cellulose, nucleopore and teflon filters are preferred over the EPA recommended fiberglass filters. Special care must be given all phases of sample collection to avoid contamination of the filters.

The method of analysis is state-of-art. Care must be given to the sample collection to insure that quality assurance practices are followed.

APPENDIX A-2

STANDARDS FOR BERYLLIUM

Montana.	0.01 ug/m ³ , 30 day average
Idaho.	0.01 ug/m ³ , max. 24 hr. concentration, not to be exceeded more than once a year.
New Mexico.	0.01 ug/m ³ based on a 30 day average
New York.	0.01 ug/m ³ (monthly average)
U.S. Federal.	(under hazardous compounds) 0.01 ug/m ³ 24 hr. average.
Israel.	0.01 ug/m ³ , not to be exceeded more than one per cent of the time in a year.

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MAY 1 1978

INTRODUCTION & SUMMARY

Lead is a gray-white metal found in relative abundance in the earth's crust. Although it occurs naturally in living organisms, lead has no biologically beneficial effect on plants, animals, or humans. It is toxic, affecting the liver, kidney, central nervous and reproductive systems. Children and pregnant women are especially susceptible. MAY 1 5 1978

Under natural conditions lead enters living organisms through the breakdown of lead-bearing minerals. Natural lead cycling in the environment includes: 1) incorporation into the upper layers of soil; 2) absorption by plants; 3) leaching into surface and ground water, or erosion, producing air-borne dust. An additional increment enters the air via the decay of radon, and the action of forest fires, sea salt, and volcanoes, (National Academy of Science, (NAS) 1972).

Background levels for atmospheric lead have been measured as low as 0.0012-0.1029 ug/m³ in California's White Mountains (Chow, 1972). Shacklette (1971) found a median level of soil lead throughout the U.S. to be 15 ppm. Water also has a natural lead content with a global mean between 1 and 10 mg/liter (Livingstone, 1963).

Because of its low melting point (321.4°C), malleability, poor electrical conductive properties and resistance to corrosion, lead has been used in a myriad of applications since early times. An indication of the increased use of lead through time is seen in the analysis of lead concentrations of annual ice layers from the interior of Greenland (Murozumi, 1969). Analysis of these layers indicates an increase from less than 0.0005 ug/kg of ice in 800 BC to more than 0.2 ug/kg in AD 1965. This 400-fold increase resulted from increases in airborne lead concentrations.

Currently world lead production exceeds 1.2 million metric tons per year (NAS, 1972). Man-generated lead and its compounds may enter the environment at any time throughout its mining, smelting, processing, use, and disposal. Table 1 estimates the lead emissions by source category for the U.S. in 1975.

In Montana there are both mobile and stationary lead emission sources. The environmental consequences to Montana residents are directly related to the quantity and pervasiveness of these sources.

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TABLE 1

ESTIMATED ATMOSPHERIC LEAD EMISSIONS FOR THE UNITED STATES, 1975*

Source Category	Annual emissions, metric tons/yr	Emissions as percentage of total
Waste oil combustion	10,430**	6.5
Solid waste incineration	1,630**	1.0
Coal combustion	400	0.2
Oil combustion	100	0.1
Gray iron production	1,079**	0.7
Iron and steel production	844**	0.5
Secondary lead smelting	755**	0.4
Primary copper smelting	619	0.4
Ore crushing and grinding	493	0.3
Primary lead smelting	400	0.2
Other metallurgical	272	0.2
Lead alkyl manufacture	1,014**	0.6
Type metal	436	0.3
Portland cement production	313	0.2
Pigments	112	0.1
Stationary source subtotal	19,225	
Mobile Source Gasoline combustion	142,000	
Miscellaneous	327	0.2
Total	161,225	100

*From Control Techniques for Lead Air Emissions, U. S. Environmental Protection Agency, 1977 in: Air Quality Criteria for Lead, Environmental Protection Agency, 1977.

**No sources in Montana.



LEAD POLLUTION IN MONTANA

A. Sources and Emissions

1. Mobile sources - From Table 1 it is evident that motor vehicles powered by gasoline containing lead additives represent the greatest source of lead to the atmosphere. MAY 1 1978

Lead is used in vehicular gasoline as alkyl lead to improve the antiknock quality of the fuel. The amount of lead in gasoline varies from 2-4 g/gal (Ewing and Pearson, 1974). Hirschler and Gilbert (1964) concluded that lead discharged to the atmosphere varies with driving speed, conditions, vehicle age, and the fuel employed. Approximately 70-80% of combusted lead eventually will be released to the atmosphere. The remainder is deposited in the motor oil. Research by Ter Haar (1972) placed lead emissions from motor vehicles at 25 ug per kilometer traveled. Cantwell, et al (1972) put emissions at 65 mg per km. These results show that motor vehicles burning leaded gasoline are significant lead emitters.

Most lead pollution from cars is emitted as particulate lead chlorobromide (PbCl₂Br). Many other compounds of less significance also are emitted. The chemical and physical transformations of lead in the atmosphere are not clearly understood and subject to much debate (Smith, 1976).

Hirschler and Gilbert (1964) suggest a wide range of particle sizes extending from 0.01 microns to 1-2 millimeters. Their study found that under city driving conditions 4-12% of the emissions were 1 micron or less while 50-67% were 5 microns or less. Muller (1963) found that under constant cruise speed, particle size decreased, with 62-80% 2 microns or less, and 68% of these less than 0.3 microns.

Gardner (1967) has shown a high correlation between lead levels in the air with gasoline consumption and traffic volume. Daines, et al (1970) demonstrated that this association was limited to a rather narrow zone

bordering the highway. The extent of this zone depends on particle size, with 85% of the lead in the air between 9 and 533 meters of roadways consisting of particles under 4 microns, with 65% under 2 microns. Still smaller particles can be transported much farther from transportation networks with consequent accumulation and environmental impacts. These impacts may include increased lead levels in soil, vegetation, water systems, and air. Numerous studies have been reviewed by Smith (1976) and show in general that 10 meters from heavily travelled highways lead averages 5-15 times background levels in soils and 50-200 times natural levels in and/or on vegetation. MAY 1 1978

In Montana there are over 11,000 miles of highways and 704,000 motor vehicles. Smith (1976) indicates that higher than normal lead concentrations are distributed in a band 50 meters on either side of roadways. If this is applicable in Montana, then 437,000 acres encompassing agricultural land, forest and densely populated areas, are subject to potential impact from mobile source lead. This estimate does not take into account off-road vehicles such as four-wheel drives, motorcycles, airplanes, snowmobiles, and boats. The areas most likely to be impacted are near large volumes of traffic such as cities and major highways.

2. Stationary Sources - In general, emissions from controlled stationary sources will have a large percentage of their lead emissions in either the aerosol or small particle size classes (about 2 microns). The small particle concentrations result from the inefficiency of current abatement technology in the removal of such particles from gas streams. These small particles are subject to long range transport and subsequent ecosystem accumulation over a large area. Pollution controls are applied to emissions from stacks, vents, and other process outlets. Other sources such as conveyors and waste piles are largely uncontrolled. In the case of the uncontrolled sources, large particle size confines distribution to the immediate vicinity.

Lead oxide is the main lead compound emitted by stationary sources. Since sulfur dioxide also is emitted from most of these sources, there is opportunity for interaction resulting in conversions in the chemical nature of the lead compound emitted. These interactions may lead to the generation of soluble compounds and airborne particles which may be accumulated in human lungs with potential impacts on health.

DRAFT MAY 1 1978

a) Anaconda Copper Smelter

The Anaconda copper smelter is a major source of atmospheric lead pollution. Lead exists as an impurity in the copper concentrate and is liberated to the atmosphere during the roasting and smelting process. According to data obtained by the company from its stack sampling program, the average lead emission from January, 1977 through June, 1977 was 42.15 metric tons per month. In the last seven months this average has been reduced to 10.35 metric tons per month, owing primarily to increased efficiencies in their pollution control systems (AQB, 1978).

b). ASARCO Lead and Zinc Complex

The extent of lead emissions from the ASARCO facilities currently are not known. In 1972 the Environmental Protection Agency (EPA) published a report documenting the prevalence of lead pollution in the Helena valley as a result of the ASARCO emissions. Although no emissions values were given for lead, operations potentially capable of emitting lead were identified. Within the lead smelter, both sintering and smelting processes emit lead to the atmosphere. The outside storage of concentrates was felt to be a major contributor of lead emissions. The zinc sintering plant's lead emissions come primarily from the charging of the fuming furnace. For approximately five minutes during the addition of the slag to the furnace, emissions escape to the atmosphere. New pollution control equipment recently has been added to reduce total emissions. A stack sampling program will begin this summer to evaluate the efficiency of this equipment.

c). Coal-Fired Generating Plants

There currently are four coal-fired generating plants operating in Montana. They are, in order of power production, Colstrip 1 & 2, rated at 350 megawatts each; the Corrette plant, rated at 180 megawatts, and the Montana-Dakota Utilities Sidney plant, producing up to 50 megatts.

Colstrip 1 & 2 burn coal with an average lead content below 10 ug/g (Commercial Testing and Engineering Co., 1972). No stack sampling data is available for these plants but based on a report from the Bechtel Corp. (1974), manufacturers of the air pollution abatement equipment, the atmospheric emissions of lead will be 1322 lbs/year, maximum. Approximately 50 percent of the lead emissions are volatilized into a gas or aerosol, while the additional 50 percent is emitted as small particulate (Battelle, 1974).

The Corrette plant burns the same coal as Colstrip 1 & 2. There are no emissions data available for this facility and therefore, based on generating capacity alone, it is assumed to emit lead to the atmosphere in quantities somewhat lower than the Colstrip units.

The Montana-Dakota Utilities power station emission and feed stock levels of lead are unknown.

B. Ambient Data

The only measurements for lead currently being taken in the state are near East Helena and Anaconda. Monthly averages indicate that levels at the Hastie site (near downtown East Helena) exceeded the current state standard of 5 ug/m^3 for four out of the 12 months in 1977. In 1978, one out of three months had average readings exceeding the standard. Measurements taken at the Microwave and Anaconda sites do not indicate any violations.

AMBIENT LEAD DATA

*violations of state standard

No. of Readings	Locale	Site	Month-Year	Monthly-Average $\mu\text{g}/\text{m}^3$
5	East Helena	Hastie	January 1977	2.96
4	"	"	February 1977	5.61*
5	"	"	March 1977	3.32
5	"	"	April 1977	6.10*
2	"	"	May 1977	2.25
6	"	"	June 1977	2.22
5	"	"	July 1977	2.78
3	"	"	August 1977	1.26
1	"	"	September 1977	1.93
5	"	"	October 1977	4.63
4	"	"	November 1977	15.36*
2	"	"	December 1977	14.04*
3	"	"	January 1978	12.73*
9	"	"	February 1978	3.47
1	"	"	March 1978	0.64
4	"	Microwave	January 1977	0.51
2	"	"	February 1977	2.02
6	"	"	March 1977	0.04
5	"	"	April 1977	0.03
5	"	"	May 1977	0.04
5	"	"	June 1977	0.92
4	"	"	July 1977	0.03
5	"	"	August 1977	0.38
4	"	"	September 1977	0.14
4	"	"	October 1977	0.32
6	"	"	November 1977	0.35
5	"	"	December 1977	2.53
12	"	"	January 1978	2.00
10	"	"	February 1978	0.49
1	"	"	March 1978	.031
4	Anaconda	Highway Jct	January 1977	0.43
4	"	"	February 1977	0.34
4	"	"	March 1977	0.60
5	"	"	April 1977	0.25
6	"	"	May 1977	0.20
4	"	"	June 1977	0.41
6	"	"	July 1977	0.05
5	"	"	August 1977	0.39
5	"	"	September 1977	0.47
5	"	"	October 1977	0.49
5	"	"	November 1977	1.71
5	"	"	December 1977	0.65
5	"	"	January 1978	0.82
4	"	"	February 1978	0.24
5	"	"	March 1978	0.74
3	"	"	April 1978	0.06

HUMAN HEALTH EFFECTS

There are many sources of lead exposure independent of air pollution. Lead is found in paint, ink, water supplies, and fresh and processed foods. Man is the depository for lead from all these sources throughout his life.

Exposure of humans to lead from atmospheric sources is basically of two types, primary and secondary. Primary exposure consists of direct inhalation, while secondary exposure pertains to ingestion of lead deposited from the air on a medium, such as food or water. The quantities absorbed by the body are determined by a variety of conditions such as the chemical form of lead, nutritional states, metabolic activity, and previous history of exposure (EPA, 1977).

Clinical studies have shown that between 20 and 40 percent of airborne lead inhaled will be deposited in the respiratory tract (Kehoe, 1961). As much as 50 percent of this is absorbed and enters the blood stream. The remaining 50 percent is moved to the throat by the cilia in the lining of the respiratory tract. It is then swallowed into the digestive system. In children, 40 percent of the lead taken into the digestive tract is absorbed as is 10 percent in adults (EPA, 1977).

A secondary source of ingested material of particular importance in children is lead deposited in dust and soil. Lead is picked up on children's hands during play and is then ingested when they put their hands, dirt and other foreign material in their mouths. Children with pica (a disease characterized by an abnormal craving for nonfood substances) are even more susceptible to this form of lead uptake (Landrigan, et al 1976).

The absorption, distribution, and accumulation of total lead in the body is via the circulating red blood cells, and then to soft tissues (primarily liver and kidney) and bone, where it accumulates. Accumulation begins as

early as the fetal stage, where the unborn may receive lead from the mother. Lead concentrations in the skeleton reflect cumulative exposure while elevated lead levels in the soft tissue and blood reflect more recent exposures (EPA, 1977). Since lead is more harmful to animals and humans in the soft tissue than in bone, blood lead levels have been used to indicate potentially hazardous dosage.

There are a number of levels of physiological impairment associated with lead exposure. Subcellular and cellular effects associated with mitochondrial function and morphology (Goyer and Knall, 1969) and cell nuclei changes (Goyer and Moore, 1974) have been reported. Reproduction, development, hepatic, endocrine, cardiovascular, immunological, and gastrointestinal effects from lead exposures are important to an understanding of lead toxicity. They have been reviewed extensively by the Environmental Protection Agency (1977) and the National Academy of Science (1972). Probably the most important areas of interest at the organ level are lead's blood, nerve, and kidney effects. A discussion of these areas follows.

Anemia is the clinical feature of lead intoxication dealing with the hematopoietic system. Anemia results, in this case, from increased erythrocyte destruction and decreased hemoglobin synthesis. In preschool children the threshold for expression of symptoms as measured in blood lead levels is 40 ug/dl and for adults 50 ug/dl (EPA, 1977).

The effects on the nervous system range from acute intoxication and fatal encephalopathy to subtle behavioral and electrophysical changes at lower levels of exposure. Within this range, effects can be correlated with blood lead levels.

Irreversible brain damage usually occurs at blood lead levels greater than 120 ug/dl (Kehoe, 1961), although such damage has been recorded at levels less than 100 ug/dl (Smith, et al 1938). Similarly, the level

where such damage occurs in children is approximately 100 ug/dl and rarely as low as 80 ug/dl (Grant, 1938). Once acute encephalopathy develops, coma and convulsions usually follow and can lead to death within 48 hours despite medical treatment. Blood lead levels at which neurobehavioral dysfunction occurs in asymptomatic children start near 50-60 ug/dl (Betts, et al 1973).

Excessive lead exposure can result in acute and chronic renal injury in man. Clinical features of acute exposures are seen as amino aciduria, glycosuria, and hyperphosphaturia (NAS, 1972). A threshold level is not definable but these effects are usually reversible with cessation of exposure and medical treatment. Chronic damage expressed as the gradual onset of arteriosclerotic changes, fibrosis, glomerular atrophy, hyaline degeneration, and reduction in kidney size can lead to renal failure and death (Cramer, et al 1974). The threshold level for these long-term effects are not known.

In summarizing the data on human health effects, it is important to try to identify the portion of the population most susceptible to the effects from lead exposure. It has been suggested in a number of studies (EPA, 1977) that pregnant women and fetuses are more susceptible than adults under normal conditions. This increased risk from elevated lead exposures appears related to lead transfer across the placental barrier and the physiological stress placed on women by pregnancy. Table 2 summarizes the relationship between blood lead levels, observed effects, and the portion of the population affected. It is evident from these data that children are also highly susceptible. Children are more susceptible than adults primarily because of their inclination to ingest lead through their mouths, and their greater rate of gastrointestinal absorption and retention.

TABLE 2

BLOOD LEAD LEVELS VERSUS LOWEST-OBSERVED EFFECTS LEVELS*

Lowest level for
observed effects,
ug Pb/dl whole blood

Lowest level for observed effects, ug Pb/dl whole blood	Observed Effect	Population Group
10	ALAD inhibition	Children and adults
15 to 20	Free erythrocyte porphyrin elevation	Women and children
20 to 25	Free erythrocyte porphyrin elevation	Adult males
40	Increased urinary ALA excretion	Children and adults
40	Anemia	Children
40	Coproporphyrin elevation	Adults and children
50	Anemia	Adults
50 to 60	Cognitive (CNS) deficits	Children
50 to 60	Peripheral neuropathies	Adults and children
80 to 100	Encephalopathy symptoms	Children
100 to 120	Encephalopathy symptoms	Adults

*From Environmental Protection Agency 1977 Air Quality Criteria for Lead

The use of blood lead levels in determining the degree of exposure to lead generally is accepted. The EPA (1977) has summarized the many studies dealing with this question near stationary and mobile sources and concludes: 1) blood lead levels resulting from increased air lead concentrations do not necessarily parallel the range of air lead levels found in the environment. The relationship is dependent on many variables, including rate of current exposure and history of past exposure. The observed ratio between blood lead levels and air lead exposures range from $1 \text{ ug/dl} = 1 \text{ ug/m}^3$ to $2 \text{ ug/dl} = 1 \text{ ug/m}^3$, respectively. Also, there are data indicating the ratio for children may be slightly higher and that male ratios are higher than females; 2) there is general agreement that blood lead levels in humans begin to increase when soil lead levels in their surroundings are from 500 to 1000 ppm. A doubling of soil lead levels correlates with a 3-6 percent mean increase in blood lead levels; 3) long-term low-level or intermittent exposures did not always correlate well with blood lead levels.

In Montana the major study associating levels of lead in soil and air to blood lead levels was carried out near the ASARCO smelting complex in East Helena by the EPA (1972). The study showed that air lead monthly averages measured in 1969 varied from 0.4 to 4 ug/m^3 at East Helena City Hall and averaged 0.1 ug/m^3 for Helena. Lead in the soil was found to be 4000, 600 and 100 ug/g at distances of 1.6, 3.2, and 6.4 km respectively from the smelting complex. Blood lead levels in children were found to be higher in East Helena than in Helena, averaging 16.6^+ , 5.1 and 11.6^+ 10 ug/dl . No adverse health effects were noted, although blood lead levels indicated an elevated exposure.

Two additional blood lead studies have been performed by the Department of Health and Environmental Sciences (Jankowski, 1975) near East Helena and Anaconda, Montana. In the East Helena study, 92 children were analyzed

with four having blood lead levels greater than 40 ug/dl and 27 with blood lead greater than 30 ug/dl. Near Anaconda, thirty-five children were sampled, with 13 having blood lead levels greater than 30 ug/dl. Although these levels cannot be directly related to airborne and soil lead levels, it does indicate evidence of a health hazard to children in these areas.

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ENVIRONMENTAL EFFECTS

A. Odors and Visibility

There appears to be no data on the effect lead emissions in the atmosphere have on odor. There also is little literature regarding effect of lead on visibility. One paper, however, estimated that automotive lead emissions could be responsible for a 25 percent or greater contribution to visibility degradation noted near freeways (Charson and Pierrand, 1969).

B. Agriculture and Other Plants

Plants are exposed to lead through their leaves, stems, bark, or roots. Meteorological conditions such as precipitation and wind modify the amounts of lead that come into contact with plant surfaces while the morphology of the plant determines the chemical nature and type of material ultimately retained. (Heichel and Hankin, 1976).

In one experiment over 95 percent of lead particulate was washed from plants by simulated rainfall (Carlson, et al 1976). Much of the lead available to plants is in insoluble and inert forms. Little (1973) indicates that only soluble lead forms are significant to plants, since insoluble and inert forms do not penetrate leaf surfaces.

Tree bark accumulates lead and has been used as an indicator of lead accumulation in ecosystems. Rolfe (1974) has shown a five-fold increase of lead levels in tree bark over a 50-year period in central Illinois.

The root system is the major avenue of uptake for lead in plants. The actual uptake of lead from soils is a function of the pH, soil sorption and cation exchange capacity (Zimdahl, 1973). Uptake has been shown to be a passive process that increases with soil lead concentration to a certain level. It also depends on soil pH, is relatively insensitive to metabolic inhibitors, and shows no unusual temperature effects (Arvik and

Zimdahl, 1974). Once uptake has occurred in root systems, lead appears to be bound primarily in dictyosome vesicles and subsequently incorporated into the cell wall with only small amounts translocated to other parts of the plant (Malone, 1974).

Most of the studies dealing with lead toxicity in plants have been carried out in artificial nutrient cultures. The literature conflicts as to where plant toxicity begins but points out the importance of such factors as physical and chemical/soil characteristics in determining the actual levels (Zimdahl, 1976).

Field experiments have not shown plant toxicity from lead pollution alone. The lack of positive results appears to be the result of either 1) environmental concentrations of lead at levels not high enough to cause observable effects; or 2) lead binding systems in the soil precluding availability to plant systems (Zimdahl, 1976). However, near highways and smelters where there has been soil disturbance, there may be a real problem with this second possibility. Taskey (1972) has described the relationship between elevated levels of heavy metals including lead and the absence of plant communities near the Anaconda smelter. The soils near Anaconda have been seriously eroded over the years, possibly leading to chemical alteration of soil lead and making toxic levels of lead available for uptake by plant communities.

Despite the many apparent impacts of lead pollution on plants and soil, the major concern is for the consequent effects on humans. Many experiments near smelters (EPA, 1972) and highways (Shuck and Locke, 1970) show increased levels of man-generated lead in and/or on foliage and consumer crops. If these plants are eaten without sufficient washing, serious health hazards could result.

C. Livestock and Other Animals

Lead poisoning to domestic animals is a problem frequently found near smelters, mines, and industrial plants (Schmidt, 1971). Although such poisoning is also a possibility near highways, no literature is available on the subject.

The effects of lead on biological processes of animals generally includes the nervous and hematopoietic system and kidneys (EPA, 1977).

Hammand and Aronson (1964) estimated the minimum cumulative dose for lead poisoning in cattle to be six to seven mg/kg body weight per day.

For horses, the toxic dosage is estimated to range from 1.7-2.4 mg/kg body weight per day (NAS, 1972). Besides having a physiological susceptibility, horses are vulnerable to lead poisoning because of their close-cropping feeding habits. Horses sometimes pull up plants and eat roots and soil along with foliage, thereby ingesting any lead present (Aronson, 1972).

A study by the EPA (1972) near the ASARCO smelter at East Helena showed that a postmortem of one horse from the vicinity indicated acute lead poisoning. One lung showed evidence of chronic lead or cadmium exposure. Pneumonia and heart disease further suggested heavy metal exposure. Gordon (1968) said it has been impossible to raise horses near the smelter because of excessive lead in the environment.

The exposure of small mammals near highways has been studied (Jeffries and French, 1972; Williamson and Evans, 1972). Both studies point to a parallel between total body lead and distance from source. In most cases, body lead and soil lead also were correlated. No lead poisoning was reported in either study.

effect on some types of electric circuits, but this has not been completely substantiated (NAS, 1972).

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Measurements of lead levels in pigeons (Tansy, 1970) and starlings (Martin, 1972; Martin and Nickerson, 1973) indicate that urban birds have higher lead levels than rural birds. Toxic dosage levels for these birds currently are not known. Toxicity associated with waterfowl feeding in areas of lead mine wastes (Chupp and Dalke, 1964) have been reported. Waterfowl appears to be twice as sensitive to the bio chemical effects of lead as man and other mammals (Finley, et al 1976).

D. Ecosystems

The effects of lead on ecosystems is implicit in the nature of lead cycling in nature. Getz, et al (1977), estimated the transportation, uptake, and movement of lead in a watershed ecosystem near and surrounding an urban center in southern Illinois. This research indicates that 92.5 percent of lead that enters the ecosystem remains within it. The major reservoirs were soils, plants and stream sediments. The major portion of the accumulated lead was in urban sectors and areas adjacent to high use rural roads. Jennett, et al (1977), attempted a similar investigation near a mining and smelter area in southeastern Missouri. This study found elevated concentrations of lead in soils, vegetation and forest floor litter with storm runoff in streams being the major transport mechanism out of the study area. Stream sediments did not concentrate lead because of water velocity but lake sediments at the stream's mouth increased in lead content.

E. Materials

Material destruction by airborne lead does not appear to be a problem of any significance. Catalytic converters in automobiles are made inoperable by lead in automobile emissions. This necessitates the use of unleaded gasoline in these cars. There is also some evidence that lead has a harmful

In 1977, the Montana Air Quality Bureau recommended to the federal government an ambient air quality standard for lead of 2.0 ug/m^3 . The recommendation was based on a review of the Yankeel and von Lindern study and an assessment of the state's emission sources and health studies.

Subsequently, the EPA (Federal Register, 1977) proposed a new ambient air quality standard for airborne lead. The proposed standard is 1.5 ug/m^3 , based on a monthly average. This is over three times as stringent as Montana's present standard of 5.0 ug/m^3 . EPA holds that the level proposed is necessary to protect the health of the more sensitive portion of the population (children) with a margin of safety. DRAFT MAY 1 1978

The EPA is scheduled to promulgate a final standard by June, 1978. By March, 1979, the states must develop plans demonstrating how they will meet the standard by 1982. Federal law thus requires the Montana ambient air quality standard for lead to be at least as stringent as the new federal standard.

CONCLUSION

In developing a standard with an adequate margin of safety, it is necessary to identify the most sensitive segment of the population and the potential pathways for exposure. Children have been identified as physiologically the most susceptible to lead toxicity. They suffer clinical symptoms of lead poisoning at lower blood lead levels than others. Children also are exposed to lead not only from ambient air concentrations but from soil lead originating as fallout from the air. Thus, a major consideration in establishing an ambient air lead standard is the residual buildup from contaminated fallout (in dust and soil) and its effect on children.

An EPA draft criteria document (November, 1976) recommended a 5 ug/m^3 standard (Montana's present regulation). Criticisms of the document (Brodevicz, 1976) said it did not consider exposure from dust and soils in establishing its margin of safety. Rather, the document dealt only with inhalation of airborne lead.

Yankel and von Lindern (1977) provide evidence that lead concentrations in the soil are of paramount importance in determining the total lead exposure for children. This study describes a range of ambient air lead concentrations corresponding to given soil lead levels. Taken together, they predict the percentage of children exceeding a blood lead level of 40 ug/dl (anemia threshold in children). It was found that concentrations of 2.0 ug/m^3 (30-day average) would leave 95 percent of children unaffected by lead toxicity. The predictions in this study were made from statistical data interpretations and did not include a margin of safety. Yankel and von Lindern's work clearly indicates the important relationship between air and soil lead levels in the setting of ambient standards.

APPENDIX B2

LEAD STANDARDS

<u>Location</u>	<u>Remarks</u>	<u>Standard</u>
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<u>United States</u>		
Pennsylvania		5 ug/m ³ 30-day average
California		1.5 ug/m ³ 30-day avg.
Oregon		3.0 ug/m ³ arithmetic average all samples at any station during any calendar month
Montana	Not to be exceeded more than 1% of the time within 1 year	5 ug/m ³ 24-hour average
<u>Foreign Countries</u>		
Bulgaria		0.7 ug/m ³ (except tetraethyl lead) 24-hr. avg. 1.7 ug/m ³ (lead sulfate) 24-hour average
Spain		50 ug/m ³ 30-minute avg. 10 ug/m ³ 8-hour average
Israel	Not to be exceeded more than 1% of the time within 1 year	5 ug/m ³ 24-hour average 3.5 ug/m ³ (lead sulfate) 24-hour average
Italy	Not more than once within an 8-hour period	50 ug/m ³ 30-minute avg. 10 ug/m ³ 24-hour average
Romania		1.0 ug/m ³ 24-hour average
Russia		0.7 ug/m ³ 24-hour average (except tetraethyl lead) 1.7 ug/m ³ (lead sulfate) 24-hour average
Poland	Maximum admissible concentration Special areas with respect to climate	1.0 ug/m ³ 24-hour average 0.5 ug/m ³ 24-hour average
Czechoslovakia		0.7 ug/m ³ 24-hour average

APPENDIX B1
MEASUREMENT TECHNIQUES FOR LEAD

Lead - Method: Fed. Reg., Vol. 42, No. 24, Dec. 14, 1977, pp. 63083-63086

Principle - Ambient air suspended particulate matter is collected on a low background filter medium using a 24-hour sampling interval. Lead in the particulate matter is solubilized by nitric acid extraction, facilitated by ultrasonication and heat. The lead content of the sample is analyzed by atomic absorption spectrometry using an air-acetylene flame under optimum instrumental conditions at either the 283.3 or 270.0 nm absorption lines.

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Montana's AQB also will recognize any analytical method with a consistent and repeatable lower detectable limit (LDL) of 0.05 micrograms lead per cubic meter of air. However, the LDL must be documented and presented to the AQB for acceptance. The filter media must be low in lead background. Cellulose, nucleopore and teflon filters are preferred over the EPA-recommended fiberglass filters. Special care must be given all phases of sample collection to avoid contamination of the filters as lead tends to be ubiquitous.

The method of analysis is state-of-the-art. Care must be given to the sample collection to insure that quality assurance practices are followed.

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